Foreword

Ten years ago, a mysterious and yet fatal encephalitis broke out among the pig farmers in Negeri Sembilan, Malaysia. There were much confusion initially regarding the cause of the illness, and Japanese encephalitis, the commonest cause of encephalitis here, was thought to be the culprit. It soon became clear that the epidemiological, clinical and radiological features of the new encephalitis were different from those of Japanese encephalitis. Viral culture from one of the patients from Bukit Pelanduk, one of the villages in the epicentre of the outbreak area, yielded a new paramyxovirus related to the then recently discovered Hendra virus. The virus was later named after the village as Nipah virus. Initial descriptions of the virus portrayed a deadly zoonotic agent originated from bats, spread to domestic animals, amplified rapidly in pigs, and then spread readily to human handling the animals. At the height of the outbreak, even abattoir workers in the neighbouring country Singapore, which depended on Malaysia for its supply of pork, were affected, and the importation of pork from Malaysia was banned. The outbreak was brought under control only with the culling of over a million pigs – and with it the decimation of a billion dollar industry. The final count was more than 265 patients affected with 105 mortalities.

Various aspects of the infection caused by this novel virus were soon documented – clinical, laboratory, radiological features, antiviral therapy, epidemiology, pathology, prognosis and relapse. It was then thought that the outbreak occurred because of the unique combination of human activities in deforestation, intensive farming practices of combining pig husbandry with fruit farming, and unusual seasonal changes in weather, something that was unlikely to recur elsewhere.

It was therefore a surprise when Nipah virus was reported to be responsible for a series of encephalitic outbreaks that occurred almost annually in northeast India and Bangladesh. Later it was found that the Henipavirus was common among many species of fruit and insectivorous bats, which were widely distributed in a region stretching from Australia to southern China, and from Indonesia to Ghana. It was also soon appreciated that there were significant epidemiological, clinical, radiological and prognostic differences between the outbreaks in Bangladesh and that in Malaysia, though some of these were more likely to be due host factors and local cultural practices and health care service conditions. Significantly, however, the virus was far more readily transmitted from bat-to-human and from human-to-human in Bangladesh than in Malaysia, and this led to increased risk of nosocomial transmission and transmission to health care workers, such as what had happened in Siliguri, India.

The outbreaks in Bangladesh and the subsequent discovery of the widespread distributions of the virus supply new impetus, funding and the opportunities to study the virus and the related infection in more depth. New cases of Hendra infections were continuously being reported in Queensland, Australia. On the other hand, 10 years long term outcomes are now available from Malaysia. The more exciting developments, however, must be the better understanding of both the genetics and the pathophysiology of Nipah infection. This issue of the Journal reports many of the latest findings in this area as presented in the Nipah Virus Colloquium 2008. The Colloquium was held on 21, 22 October 2008 in University of Malaya, Kuala Lumpur, in conjunction with the 10th year remembrance of the outbreak and discovery of the Nipah virus. It is hope that, through efforts like these, we are better prepared for the next re-emergence of Henipaviral infection.

Heng Thay Chong
Sazaly Abu Bakar
Chong Tin Tan
Editors
REFERENCES